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Stable Ischemic Heart Disease

LIPID TRANSFER TO HDL IN PATIENTS WITH CORONARY ARTERY DISEASE

Poster Contributions
Hall C
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Background: Systematic assessment of metabolic and functional aspects of HDL is important for the understanding of HDL anti-atherogenic role and the establishment of new CAD markers and therapeutic goals. Lipid transfer between HDL and the other lipoproteins, a process mediated by CETP and PLTP, is a crucial step in HDL metabolism and determinant for HDL functions in cholesterol esterification and reverse cholesterol transfer. Our objective was to investigate whether development of CAD is associated with alterations in lipid transfers to HDL and in content of free cholesterol in the plasma and in HDL.

Methods: 71 patients with CAD (CAD) and 78 without CAD (n-CAD), as diagnosed by cineangiography, were studied. They were of both genders, aged 40-80 yrs. Fasting plasma samples were incubated for 1h at 37oC with a donor artificial nanoemulsion labeled with 3H -cholesteryl-esters and 14C -phospholipids or 14C- free-cholesterol and 3H -triglycerides. Radioactive lipids transferred from the donor nanoemulsion to HDL were quantified in the supernatant after chemical precipitation of non-HDL fractions and nanoemulsion.

Results: In CAD group, total cholesterol, LDL-cholesterol, triglycerides and apoB were higher than in n-CAD. CAD showed diminished transfer to HDL of free-cholesterol (CAD=6.9 ± 1.3; n-CAD= 8.3 ± 1.5%, P<0.0001), triglycerides (4.4 ± 0.8 vs 5.0 ±0.6%, P<0.0001), and phospholipid (21.2 ± 3.1 vs 25.4 ± 2.3%, P<0.0001), whereas the transfer of cholesteryl-ester was higher (5.0 ± 1.0 vs 4.3 ± 0.9%, P<0.0001). Plasma free-cholesterol was higher in CAD (CAD=36.8 ± 8.1; n-CAD=34.4 ± 7.1, P=0.04), while the content of free-cholesterol in HDL was smaller (7.6 ± 2.4 vs 8.8 ± 2.9, P<001). HDL particle diameter was higher in CAD (8.5 ± 0.5; 8.2 ± 0.4; P<0.0001) as well as CETP concentration (3.5 ± 0.7 vs 3.0 ± 1.0, P=0.003).

Conclusion: The reduction of free-cholesterol transfer to HDL may impair cholesterol esterification and reverse cholesterol transport. Alterations in triglyceride and cholesteryl-ester transfer may affect lipoprotein stability. Those disturbances in HDL metabolism may facilitate CAD development.